3 months of training. How significant is this as our SHOs perform bag mask ventilation on a daily basis, prior to laryngeal mask airway (LMA) insertion and tracheal intubation? It is a common criticism that (over)use of the LMA means trainees perform less intubations and I welcome the recommendation that trainees should be preferentially attached to lists where facemask anaesthesia and intubations occur. Trainees moving between theatres will also increase the number of intubations performed, but it is important not to forget the issue of extubation, another core skill for all anaesthetists and as important as tracheal intubation.

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Editor—We were interested to read the comments made in response to our study ‘A Scottish National Prospective Study of airway management skills in new-start SHOs’ and welcome the opportunity to respond to them.

Firstly, to address Dr Ratnalikar: as this was a study of trainees all over Scotland, it is not possible to comment on the type of cases being done using a facemask only in many of the workplaces included in the study. However, in our own institutions, trainees are directed to day case minor gynaecological, urological, and orthopaedic surgery to gain experience using a facemask. Some of these cases are opportunistic learning exercises, and a senior anaesthetist working alone may elect to use an LMA in place of the facemask, but can identify a training opportunity when a trainee is present.

Secondly, in response to Dr Clarence: there are many types of video-assisted aids to laryngoscopy available today. While some may have a place in clinical practice, particularly when the more difficult airway is encountered, we firmly believe that new-start trainees should master basic skills such as facemask holding and direct laryngoscopy before progressing to experiment with such adjuncts.

Thirdly, we agree with Dr Hodgetts that although trainees can move between theatres to maximize intubations performed, this is at the expense of learning the conduct of anaesthesia and the management of emergence and extubation. This period is vitally important and can be the source of many critical incidents if not managed correctly. Our personal experience is also that some Consultants find it disruptive to running a list and teaching trainees if they are popping in and out of theatre repeatedly.

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1 LMA is the property of Intavent Ltd.

Fluid responsiveness using non-invasive predictors during major hepatic surgery

Editor—We read with great interest the study by Solus-Biguenet and colleagues regarding the evaluation of fluid responsiveness using non-invasive predictors during major hepatic surgery. The authors compared the value of several indices of fluid responsiveness and concluded that the respiratory variations in arterial pulse pressure obtained from the invasive (PPVart) and Finapress™ (PPVfina) arterial pressure curves were the most accurate predictors of response to volume expansion. Moreover, they concluded that respiratory variations in the pulse oximetry waveform (PPVsat) were greater in responders than in non-responders to volume expansion even if the predictive value of this parameter was weaker than PPVart and PPVfina. These results are extremely interesting, as pulse oximeters are non-invasive, inexpensive, and are daily used in the operating theatre. Thus, the results from this study suggest that PPVsat may be a useful predictor of fluid responsiveness in the operating theatre. However, in our opinion, technical description regarding the way the pulse oximetry waveform was acquired in this study is not clear enough to sustain the hypothesis that PPVsat is a weaker predictor of fluid responsiveness than PPVart and PPVfina. The software generates a signal that is substantially filtered, amplified, and smoothed before display. However, some data acquisition software allows disengaging the automatic gain incorporated in the pulse oximeter in order to avoid the potential influence of the signal processing on the displayed waveform. Using this technique, PPVsat has been shown to be strongly related to PPVart with far lower limits of agreement than those described in the present study. We can postulate that Solus-Biguenet and colleagues did not control this limiting factor as no mention is made concerning the gain and as agreement between PPVart and PPVsat was lower than those previously reported.

As some recently published studies are suggesting, PPVsat is strongly influenced by the site of measurements (ear, finger, forehead). Consequently, it is of major importance to mention the site of measurement and to consistently use the same site between patients, as an up to 10-fold variation can occur. Mixing the sites may induce an important bias.
The authors postulate that the sensitivity of the plethysmographic signal to humoral and neurogenic factors may explain the poor predictive value of PPV\textsubscript{sat}. However, we can postulate that these potentially confounding factors are constant throughout a single respiratory cycle and that they do not impact on the minimal and maximal pulse oximeter waveform amplitudes during the same respiratory cycle. On the other hand, peripheral vasoconstriction may alter the pulse oximeter signal quality. Most monitors display a signal quality index or a perfusion index providing informations regarding the quality of the curve. These data should have been controlled before pulse oximeter waveform recording and analysis in order to avoid confounding factors related to poor signal quality.

Pulse oximeter waveform is influenced by outside light absorption. Thus, the pulse oximeter should be wrapped in order to prevent outside light from interfering with the signal. This is not mentioned in this study.

In conclusion, pulse oximetry waveform has been shown to be strongly related to respiratory cycles in previously published studies. These variations have been shown to be related to PPV\textsubscript{art} and to loading conditions. However, this waveform depends on signal processing, site of measurement, peripheral vasoconstriction, and outside light absorption. This study shows promising results regarding the ability of PPV\textsubscript{sat} to predict fluid responsiveness. We can postulate that further studies without automatic gain control, standardized site of measurements, and adequate signal quality index and recording will improve the predictive value of this new index.

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Editor—We appreciate Dr Cannesson’s interest in our recent publication. While we found that the respiratory variation in the pulse oximeter waveform (quantified by the PPV\textsubscript{sat}) predicted fluid responsiveness less accurately than either the respiratory variation in radial artery pulse pressure (PPV\textsubscript{art}) or the non-invasive PPV obtained using the Finapres\textsuperscript{TM} device (PPV\textsubscript{fina}), Dr Cannesson suggests that technical problems in acquiring or processing the signal from the pulse oximeter waveform may be responsible for this lack of reliability.

Dr Cannesson first states that avoiding the potential influence of the signal processing on the displayed waveform by disengaging the automatic gain incorporated in the pulse oximeter may allow obtaining a stronger relation between PPV\textsubscript{sat} and PPV\textsubscript{art}, and postulates that we did not control this limiting factor. This, however, cannot be an explanation for our results, because we actually used a manual gain control in our study. Moreover, a change in gain between two sequences of measurements would not change the value of PPV, because PPV is a relative indice (the difference between minimum and maximum pulse waveforms within a respiratory cycle divided by the mean of the two values). This can be easily verified by changing the gain between two successive waveform recordings. In addition, it should be mentioned that auto-centering algorithms, which are not deactivated when automatic gain is disengaged, rather than gain itself, are likely to alter the effects of ventilation on the waveform.

Dr Cannesson cites the crucial importance of using only one site of measurement, because this has been shown to strongly influence the value of PPV\textsubscript{sat}. Dr Cannesson even states that PPV\textsubscript{sat} may be more than 10 times stronger in the region of the head when compared with the finger. This is an over-interpretation of the results of a study which quantified the amplitude of the power spectrum at the respiratory frequency following spectral analysis of the waveforms, not PPV\textsubscript{sat} itself, which quantifies only one component of the waveform variations induced by ventilation. Interestingly, this study strongly supports the notion that pulse oximetry cannot be recommended to accurately assess the respiratory variation in arterial pressure. Nevertheless, it is true that the whole waveform, including the respiratory change in its pulsatile component, and thus PPV\textsubscript{sat}, may vary in a given patient with the site of measurement. We have anecdotaly observed in patients with septic shock that the effect of ventilation on the pulse oximeter waveform could even be dramatically different between two fingers of the same hand (data not shown). In our study, measurements in a given patient were always performed at the same finger, and never at the ear or forehead location.

Dr Cannesson also postulates that because they are constant throughout a single respiratory cycle, humoral and neurogenic factors should not significantly impact the minimal and maximal waveform amplitudes, and thus PPV\textsubscript{sat}, during the same respiratory cycle. We agree that this hypothesis may be true, although, at this time, it remains to be demonstrated. In fact, the clinical evidence for this to be true comes from our results obtained not with PPV\textsubscript{sat}, but with PPV\textsubscript{fina}, since the Finapres\textsuperscript{TM} device also measures arterial pressure at a distal site and predicted fluid responsiveness as accurately as PPV\textsubscript{art}.

Finally, Dr Cannesson proposes that the pulse oximeter waveform should be used only when perfusion is good enough (as attested by the signal quality index of the monitor), the gain adequately controlled, the site of measurement unique, and the probe wrapped (to prevent outside light from interfering with the signal). In our study, poor signal or perfusion was explicitly recognized as a potential limitation of both PPV\textsubscript{fina} and PPV\textsubscript{sat}. Measurements were performed throughout major surgery, and reduced finger perfusion may in part account for the lack of agreement between PPV\textsubscript{sat} and PPV\textsubscript{art}. We feel that one of the most interesting results in our study was precisely that, in similar experimental conditions (of
peripheral perfusion, temperature, light exposure...), PPViina was a better predictor than PPVsat. These conditions are indeed different of those advocated by Dr Cannesson, but likely represent 'real life', when anaesthetists wonder whether fluids should be given to their patient in the operating theatre. We agree with Dr Canesson that PPVsat, in these conditions, may be of some value and, accordingly, our study showed that PPVsat was a better predictor than the classical static pressure measurements. Nevertheless, in accordance with other reports,3 6 our results also suggest that PPVsat should be used with caution in this indication. From a theoretical point of view, it is conceivable that modifications in device algorithms may allow commercial pulse oximeters to provide results similar to those obtained with the Finapres™.

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Changes in Doppler velocimetry of uterine arteries during labour analgesia

Editor—I was interested to read the study by Chen and colleagues1 in which they measured uterine artery resistance during the course of labour and after delivery in 20 women who received epidural analgesia. The paper is called ‘The effects of continuous epidural analgesia on Doppler velocimetry of uterine arteries during different periods of labour analgesia’ and concludes ‘Continuous epidural analgesia with bupivacaine 0.075% increases the resistance of uterine artery and therefore possibly reduces uterine blood flow.’ This conclusion is, on the face of it, unlikely, given the probability of some sympathetic blockade affecting the region. Moreover, there is no way such a conclusion can be drawn from this study. There were no controls who laboured without epidural analgesia. What the study showed were changes in uterine artery resistance in the course of labour and after delivery in women all of whom happened to receive epidurals.

When considering maternal changes that might affect fetal welfare for one reason or another, it is also important to assess the actual neonatal outcome. Having no controls the authors were in no position to do this, but fortunately many studies have been conducted in which the effect on neonatal outcome of neuraxial has been compared with other types of analgesia and no analgesia. Meta-analysis has demonstrated clear neonatal benefit associated with epidural analgesia in improving Apgar score6 and, more importantly, reducing metabolic acidosis,7 the latter even in comparison to no analgesia.4

It is crucially important that such spurious adverse findings do not get into the hands of the many who campaign to minimize epidural use in obstetrics, thereby depriving mother and baby of the benefits that we as anaesthetists quite fail to publicize.

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Editor—We would like to thank Professor Reynolds for helping elucidate the controversy in our manuscript.1 First, we agree that an additional group of parturients who laboured without epidural analgesia is the ideal control group. However, the essence of our study design was to compare Doppler velocimetric variables between uterine contraction and relaxation. All parturients must be measured sequentially along different time points (1, 2, 4 h) after epidural infusion and post-delivery. Furthermore, we also noted Doppler velocimetric measurements comparable to our pre-epidural control in a non-epidural control of Bhushan and colleagues.5 Thus, we believe that the pre-epidural insertion data (time 0) in our study design could also serve as an appropriate control.

Secondly, Dr Reynolds has precisely pointed out the limitation in velocimetric measurements of uterine artery and potentially misleading interpretation in our discussion. As with most previous investigations, we used RI, S/D ratio and PI to measure uterine artery resistance. Neither uterine nor umbilical blood flow was measured. Thus, our evidence